Our ambulance crews are in an extremely good position to give very valuable information to the casualty staff about the conditions in which they found the patient and the history as obtained from neighbours, friends, and relatives of the patient. In this particular case the ambulance crew had clearly brought the patient from her house to the hospital and, I suspect, as so often happens left the department without anyone recording the information that they had about where the patient was. If she had, indeed, been found naked on the floor in a bathroom, this information should have alerted the hospital staff to the possibility of a head or neck injury.

As we are training our ambulance crews to a higher and higher standard, it is vital that their initial observations, examination, and treatment of the patient are recorded and that a copy of their recorded details are filed in the hospital notes. This has been something that the British Association for Immediate Care has been studying and campaigning for over a long period, and it is high time that casualty surgeons and others recognised this need and that immediate steps be taken to implement it.

K C HINES

London E18 2AD

The father of modern surgery

SIR,—I enjoyed Mr J A Shepherd's biography of Lawson Tait, which was recommended by Professor Harold Ellis (1 May, p 1316). In support of his title "the father of modern surgery," certain aspects of Tait's career merit amplification.

During a relatively short surgical practice Tait was credited with an unparalleled 5000 abdominal "sections," performed when many surgeons viewed opening the peritoneum as extreme recklessness. Though he pioneered many operative procedures, surely his main contribution was the demonstration that abdominal surgery could be safe. His adopted technique was simplicity itself—a ligature. Until then there was no satisfactory method of dealing with a vascular pedicle as ligatured stumps usually become septic, leading to peritonitis, haemorrhage, and death. The practice of leaving the ligature ends long through the wound relied on thrombosis prior to their separation. Spencer Wells introduced a vascular clamp for ovariotomy, which was also brought out externally and removed around the fourth postoperative day. His "low" mortality of 25% brought him fame, and a modified clamp bearing his name is in general use today. Tait cut his ligature short and published astounding results, including one series of 139 ovariotomies without death. An explanation of his success is as simple as his ligature technique itself—he boiled the ligatures. He had no understanding of "germs," and repeatedly criticised Lister's antiseptic method, yet unwittingly he had stumbled upon aseptic surgery.

Tait's career came to an undignified and abrupt end. The major factor was the allegation that he had fathered a child to one of his nurses, allegations which were never proved. Even in North America, where he had more followers than at home, his reputation was blighted by "libellous statements" made by the then editor of the *British Medical Journal*, Dr Ernest Hart. Though Victorian society found favour with a surgeon with controversial methods, there was no place for a surgeon with questionable morals. Tait resigned from the Birmingham Women's Hospital in 1893. His practice declined, and he died almost forgotten in 1899. We should, indeed, be grateful to Mr Shepherd for bringing Tait to life again.

J A C BUCKELS

Walsgrave Hospital,
Coventry CV2 2DX

The severely ill child

SIR,—I would like to make a comment about the article by Dr H B Valman (8 May, p 1388) in his series on the ABC of 1 to 7. In the discussion about severe dehydration he makes no mention of hypernatraemia, which requires an altogether different method of management from other types of dehydration. The incorrect management of this problem can lead to severe neurological sequelae. I think it is important that general practitioners are aware of the condition and also that medical staff dealing with acutely ill children are aware that fluid replacement is very critical and fluid loss should not be replaced quickly.

MICHAEL L SMITH

Leeds Road Hospital,
Bradford,
West Yorkshire BD3 9LH

**This rare problem was considered in detail in a previous article on diarrhoea. It was omitted from the last paper as a considerable amount of material had to be compressed. The problem of hypernatraemia has, however, been included in the book which will be published shortly.—ED, *BMJ*.

Self-poisoning with sustained-release aminophylline: secondary rise in serum theophylline concentration after charcoal haemoperfusion

SIR,—We read with interest the case report by Dr J M C Connell and colleagues (27 March, p 943) in which they noted a secondary rise in serum theophylline concentration after charcoal haemoperfusion. While we accept that this rise in part is due to redistribution of drug from tissues into plasma, we would disagree that such redistribution carries the risk of "serious rebound drug toxicity." Additionally, however, we do not feel that the authors have adequately excluded continuing theophylline absorption as the mechanism underlying the "secondary rise" in serum concentration.

We have previously shown that the pharmacological effects of theophylline relate to its concentration in a peripheral compartment rather than in the central—that is, plasma—compartment,[1] and it, therefore, seems likely that toxic effects will similarly depend on the theophylline concentration in the peripheral compartment. The "secondary rise" in serum theophylline concentration may therefore represent *reduced* toxicity (since drug is leaving the peripheral compartment). In addition, the rapid fall in serum theophylline due to haemoperfusion may not in fact represent reduction in the risk of toxicity because drug is removed primarily from the central compartment.

Although a secondary rise in serum theophylline concentration is a well recognised phenomenon, we suggest that the magnitude of the rises seen in this case (18 mg/l and 19 mg/l) are too great to be attributable to redistribution alone. In pharmacokinetic terms such redistribution depends on the rate constants applied between the peripheral and central compartments. The mean value in the literature for the rate constant (k_{21}) between the peripheral and central compartment is 3·67 hours^{-1} (range 2·21-4·68). We calculate that it would require a rate constant of less than 1% of that value before redistribution changes of about 18 mg/l could be achieved, and we, therefore, suggest that it is most unlikely that redistribution is the sole mechanism. Instead, continuing drug absorption seems to be a more likely explanation (as the authors concede for the first period of haemoperfusion).

Thus, we fully agree with Dr Connell and his colleagues that prolonged monitoring is very important in such cases, but we do not think that they have adequately demonstrated that serious rebound toxicity occurs. There is, therefore, no hidden danger with charcoal haemoperfusion, and clinicians should persist with this therapy until plasma concentrations stabilise within the therapeutic range.

A D STRUTHERS
HENRY L ELLIOTT
G J ADDIS

University Department of Materia Medica,
Stobhill General Hospital,
Glasgow G21 3UW

[1] Whiting B, Kelman AW, Barclay J, Addis GJ. *Br J Clin Pharmacol* 1981;12:481-8.

Problems with perinatal pathology

SIR,—As a retired perinatal pathologist, now re-employed I applaud Dr A J Barson and his colleagues (27 March, p 973) for drawing attention to the present critical situation in perinatal pathology. I share, however, Dr D I Rushton's doubts (24 April, p 1265) about the feasibility of the remedies proposed.

Enough has been said to show that involving the coroner in every single case of perinatal death is neither practicable nor, indeed, desirable. The establishment of a chair in perinatal pathology would be welcome because it would raise the prestige of this subspecialty, but it would make an impact on present-day difficulties only if it could also serve as a centre for training future specialists.

Dr Barson and others recall the days when general pathologists used to do an appreciable number of perinatal necropsies but do not seem to remember that with few exceptions babies' necropsies were unpopular in general pathology departments and often disposed of in a cursory way. In some places they were regularly delegated to the most junior members of the staff.

In the years that have passed since Potter's (1952)[1] and Morison's (1952)[2] textbooks first appeared (roughly also the period of my own commitment to this discipline) the number of perinatal deaths—and presumably of necropsies—has greatly diminished, but this has been offset by greater depth. The expertise expected and required in the performance of a perinatal necropsy has increased, outstripping similar developments in other branches of histopathology. One reason for this is the virtual elimination of some of the readily diagnosable causes of perinatal death, in particular haemolytic disease of the newborn and traumatic intracranial haemorrhage. Similarly intrapartum anoxia, which in 1958[3] topped the list of causes of perinatal loss, has all but disappeared from the postmortem table. In consequence a relative increase in "exotic" causes of perinatal death necessitates not only more extensive know-